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Leucophores of the Dark-banded Rockfish *Sebastes inermis* I. Adrenergic Mechanisms that Control the Movements of Pigment

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ABSTRACT—There are unusual chromatophores, which appear yellowish in reflected light, in the integument of the dark-banded rockfish *Sebastes inermis*. Earlier ultrastructural observations revealed that these chromatophores are leucophores with granular pigment organelles [13]. In the present experiments, adrenergic mechanisms controlling the migration of pigment associated with these chromatophores were investigated. K^+ ions induced dispersion of the pigment. The dispersion was prevented by propranolol, an adrenergic blocker, and was absent from denervated preparations, suggesting that the chromatophores are innervated by adrenergic nerves. Adrenergic agonists, namely, isoproterenol and norepinephrine but not epinephrine, induced the dispersion of pigment. Propranolol inhibited the dispersion response whereas yohimbine did not block the response. Both forskolin and 8-Br-cAMP were effective in dispersing the pigment. Epinephrine was effective in accelerating the aggregation of pigment and this effect was inhibited by yohimbine. These results indicate that stimulation of beta-adrenoceptors results in an intracellular increase in levels of cAMP which then initiates the dispersion of pigment and that alpha-adrenoceptors mediate the aggregation of pigment. These adrenergic mechanisms are similar to those associated with leucophores in *Oryzias*. These findings demonstrate that the novel chromatophores behave physiologically as a type of a leucophore.

INTRODUCTION

Light-reflecting chromatophores in poikilothermic vertebrates are generally classified into leucophores and iridophores by reference to their morphological and physiological characteristics. Leucophores are dendritic in shape and contain numerous light-scattering granules, the leucosomes, which migrate centrifugally and centripetally within the cells in response to various stimuli. By contrast, the iridophores are variably in shape and contain organelles that resemble flat platelets. These organelles are usually arranged in highly oriented stacks and function as reflecting platelets.

Leucophores have attracted the attention of many researchers because, among other reasons, their movements in response to various stimuli are the opposite in terms of direction of those found in other pigment cells. However, available information on leucophores in poikilothermic vertebrates is limited to a few species of fish. Ultrastructural studies have been performed on the cells of *Fundulus heteroclitus* [22], *Lebistes reticulatus* [30] and *Oryzias latipes* [17, 26]. These studies have revealed that the structure of the leucosomes is somewhat variable, even in these fish species. To date, most of our physiological and pharmacological information about leucophores has been obtained exclusively from *Oryzias latipes*. Therefore, comparative studies on leucophores are needed for a complete understanding of the general problem of their regulation, since we know that the mechanisms that regulate the movement of chromatophores, even in melanophores, are diverse [4].

It was assumed for many years that iridophores are non-motile. In the 1980's, iridophores that participated actively in color changes, namely, motile iridophores, were reported in several fish

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species [12, 19, 28]. These motile iridophores are classified into two types on the basis of their pattern of movements: the damselfish type, in which the motility involves changes in distances between adjacent platelets in piles of platelets; and the goby type, in which platelets migrate within cells [21]. Our initial studies on motile iridophores revealed the regulatory mechanisms differ characteristically between iridophores and leucophores [15, 20]. These results stimulate us to perform comparative studies of light-reflecting chromatophores.

Recently, we found unusual light-reflecting chromatophores, which appeared yellowish in reflected light, in the integument of the dark-banded rockfish, *Sebastes inermis.* These chromatophores were motile and their responses were the opposite of those of melanophores. Ultrastructural observations revealed that these chromatophores were a type of leucophore [13].

The present report describes the adrenergic mechanisms that control the movements of pigment in these chromatophores. Our results indicate that, physiologically as well as morphologically, these chromatophores are indeed a type of leucophore.

MATERIALS AND METHODS

The dark-banded rockfish, *Sebastes inermis,* of body length of 7–10 cm (the young form) was employed. The fish were collected off the coast of the Shimane peninsular in Shimane Prefecture and they were reared in a seawater aquarium in our facilities for at least three days for acclimatization.

Isolated scale preparations were employed for physiological experiments. In some experiments, scales from chemically sympathectomized fish were employed as denervated preparations. The denervation was achieved by intraperitoneal injection of 80 μg/g body wt. of 6-hydroxydopamine hydrobromide (Sigma Chemical, St. Louis, MO) as described elsewhere [14]. Scales were carefully pulled out from the dorso-lateral part of the trunk. Each isolated scale was attached, epidermal side down, under a coverslip,which was mounted in a small chamber filled with a physiological saline solution for marine teleosts, which had the following composition (in mM): NaCl, 223; KCl, 8.1; $CaCl_2$, 2.7; $MgCl_2$, 3.7 and Tris-HCl buffer, 5.0 (pH 7.4).

Responses of leucophores were photographed under the light microscope. The magnitude of each response was calculated, from the photographs, as the change in the length of a given process in a cell [8].

The drugs used were as follows: *l*-norepinephrine hydrochloride; *l*-epinephrine bitartrate; *l*-isoproterenol hydrochloride; yohimbine hydrochloride; *dl*-propranolol hydrochloride; forskolin and 8-bromoadenosine 3′,5′-cyclic monophosphate sodium salt (8-Br-cAMP). These drugs were obtained from Sigma Chemical. Stock solutions of these drugs were diluted with physiological saline immediately before use.

For experiments on nerve stimulation, a K^+-rich saline solution was employed since the responses of melanophores and of leucophores also to K^+ ions are known to be induced *via* the release of neurotransmitter from sympathetic fibers [3, 8]. For the K^+-rich saline, we used a mixture of equal volumes of an isotonic solution of KCl and physiological saline [8].

All experiments were performed at room temperature (20–25°C).

RESULTS

General description of leucophores of Sebastes inermis

In the dermis of an isolated scale, we observed four kinds of chromatophore: melanophores; erythrophores; and two kinds of light-reflecting chromatophore. One kind of light-reflecting chromatophore was the iridophore, which appeared an iridescent bluish white in reflected light. These iridophores were non-motile. Another kind of chromatophore was dendritic in shape and appeared a dullish brown in transmitted light but yellowish in reflected light. These chromatophores were the focus of the present investigation.

In an equilibrium state in physiological saline, the chromatophores of interest appeared punctate, but they became dendritic in response to an appropriate stumulus, such as norepinephrine.

Thus, the cells are typical motile cells. Since these chromatophores are morphologically a type of leucophore [13], they are referred to as leucophores hereinafter for convenience.

The leucophores were randomly distributed, with no particular spatial association with melanophores, unlike the leucophores of *Oryzias latipes* which are found just under the melanophores. The diameter of a single cell varied from 60 to 140 μm. Figure 1 shows the leucophores in a scale of *Sebastes inermis* under reflected light.

Responses to K^+ ions

When K^+-rich saline was applied to leucophores in a punctate state in physiological saline, the leucophores responded by dispersion of the pigment within the cells. The response proceeded slowly after a lag period of about 5 min and about 30 min were required for full dispersion. When cells were returned to physiological saline, the pigment in the leucophores slowly began to aggregate. The time required for recovery of the initial pigmentary state was usually more than 1 hr, or 40 min at the very least (Fig. 2). The response to K^+ ions could be induced repeatedly in a single cell.

Melanophores in the same preparation responded to K^+ ions by the rapid aggregation of melanosomes, as do those in many teleost fish. Thus, the directions of the response were opposite in melanophores and leucophores. Figure 3 shows a typical result for melanophores in an isolated scale of *S. inermis, in* which the response was recorded photoelectrically [10]. The melanophores began to respond to the stimulus by aggregation of pigment within 20 sec and full aggregation was achieved within 3 min. When the

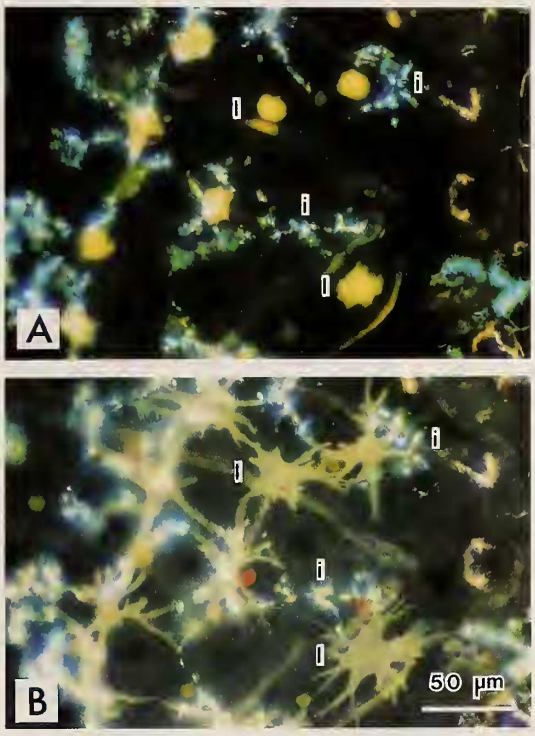

FIG. 1. Photomicrographs showing leucophores in a scale of *Sebastes inermis* (dark-field epi-illumination optics). A: Equilibrated in physiological saline. Pigment granules are aggregated in the cells. B: Pigment-dispersed state, 40 min after the application of 3×10^{-5} M norepinephrine. l, Leucophores; i, Iridophores.

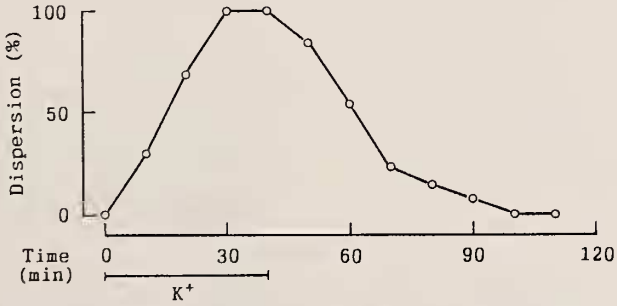

FIG. 2. Typical recording showing the dispersion response of leucophroes to K^+-rich saline (K^+) and the recovery response in physiological saline solution.

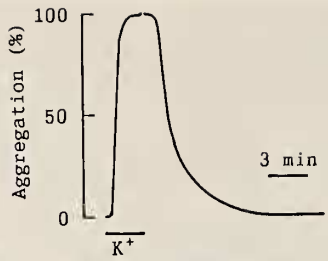

FIG. 3. Typical recording showing the aggregation response of melanophores to K^+-rich saline (K^+) and the recovery response in physiological saline solution.

melanophores were retured to physiological saline, the initial state of the pigment was restored within 5 min. The observations demonstrate that the slowness of the response of the leucophores was dependent on the motility of the cells themselves, and not on the slowness of the diffusion of experimental solutions to the place at which pigment cells were distributed.

To confirm that K^+ ions stimulate the chromatic nerves, the effect of these ions was examined in denervated preparations. No effect was apparent, not only in the leucophores, but also in the melanophores, in denervated preparations. The leucophores in such preparations did, however, respond by dispersion of pigment to norepinephrine (Fig. 4).

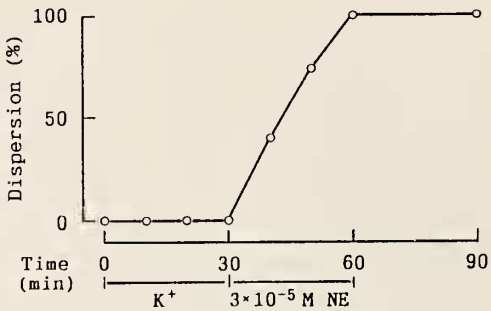

FIG. 4. Typical recording showing responses of denervated leucophores to K^+-rich saline (K^+) and norepinephrine (NE).

Effects of adrenergic antagonists on the K^+-induced dispersion response

The results of the above experiment showed that the leucophores were innervated and also suggested that the innervating nerves might be adrenergic. In this experiment, therefore, the nature of the nerve was examined pharmacologically. Yohimbine, an alpha-adrenergic blocker, did not interfere with the action of K^+ ions (Fig. 5A), while propranolol, a beta-adrenergic antagonist, effectively inhibited the action of K^+ ions (Fig. 5B). These results indicate that the nerves that controlled the leucophores were adrenergic and that the adrenoceptors mediating the dispersion of pigment in the cells were of the beta type.

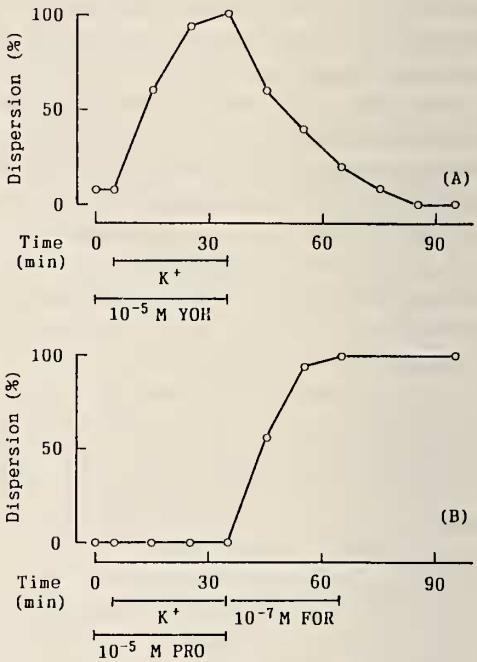

FIG. 5. Typical recording showing the effects of adrenergic antagonists on the dispersing action of K^+ ions. A: Effects of an alpha-adrenergic antagonist, yohimbine (YOH). B: Effects of a beta-adrenergic antagonist, propranolol (PRO). FOR, Forskolin.

Effects of catecholamines

To clarify the nature of the adrenoceptors that mediate dispersion of the pigment, the effects on

leucophores in an aggregated state of three catecholamines, namely, norepinephrine, epinephrine and isoproterenol, were examined. As shown in Figure 4, more than 30 min were required for maximal dispersion even at a high concentration of norepinephrine. Therefore, the magnitude of responses was measured at the end of a 60-min exposure to the drugs.

Norepinephrine induced dispersion of the pigment granules in a concentration-dependent manner. The threshold concentration of norepinephrine for an observable dispersion of the pigment granules was about 10^{-6} M, and 3×10^{-5} M was sufficient to cause full dispersion. Isoproterenol was more effective in dispersing the pigment than norepinephrine and its effect was also concentration-dependent.

Unexpectedly, epinephrine was almost ineffective in inducing the dispersion of pigment, even at high concentrations. We observed very occasionally that a few leucophores (about 5% of the observed cells) responded with half-maximal dispersion at a concentration of 5×10^{-5} M. The relationships between the concentration of each amine and the magnitude of pigment dispersion are shown in Figure 6. Isoproterenol was more effective than norepinephrine in inducing the dispersion of pigment. The EC_{50} values (concentrations that induced 50% of maximum dispersion of pigment) for isoproterenol and norepinephrine were calculated to be 9.8×10^{-9} M and 4.2×10^{-6} M, respectively.

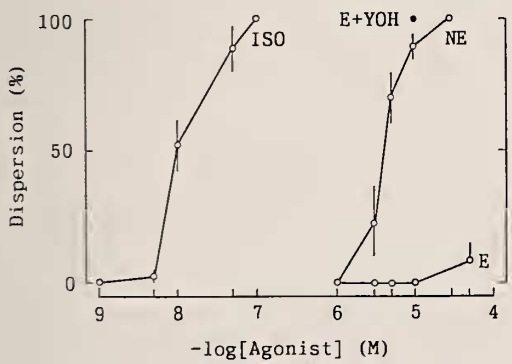

FIG. 6. Relationship between the concentration of isoproterenol (ISO), norepinephrine (NE) and epinephrine (E) and the magnitude of the dispersion response of leucophores of *Sebastes inermis*. E+YOH, Epinephrine in the presence of 5×10^{-5} M yohimbine. Each point represents the mean of 10–15 meaurements from three scales. Vartical lines indicate SD.

Effects of adrenergic antagonists on the norepinephrine-induced response

After a 5-min pretreatment with 10^{-5} M yohimbine, 10^{-5} M norepinephrine was applied in the presence of the antagonist to leucophores in a state of pigment aggregation. Yohimbine did not interfere with the effect of norepinephrine (Fig. 7A), whereas propranolol completely blocked the effect of norepinephrine (Fig. 7B).

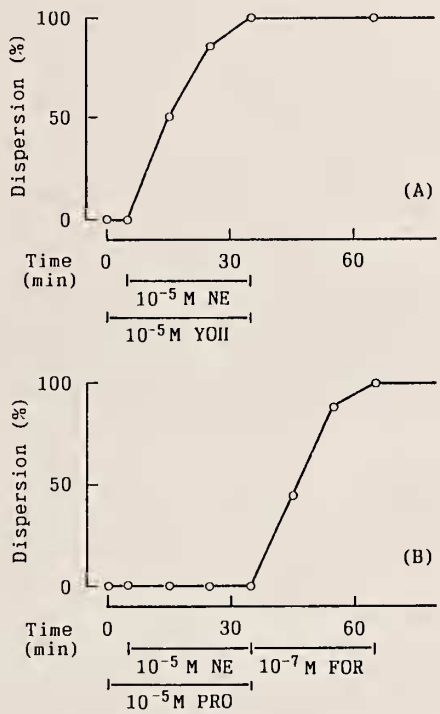

FIG. 7. Typical recordings showing the effects of adrenergic antagonists on the pigment-dispersing action of norepinephrine (NE). A: Effects of an alpha-adrenergic antagonist, yohimbine (YOH). B: Effects of a beta-adrenergic antagonist, propranolol (PRO). FOR, Forskolin.

Effects of forskolin

In order to examine the possible involvement of an adenylate cyclase system in the dispersion response of the leucophores, the effect of forskolin,

an activator of adenylate cyclase, was examined. Forskolin induced the dispersion of pigment in the leucophores (Figs. 5B, 7B). Figure 8 shows the relationship between the concentration of the drug and the extent of pigment dispersion. The EC_{50} of forskolin for inducing dispersion of pigment was estimated to be 7×10^{-9} M.

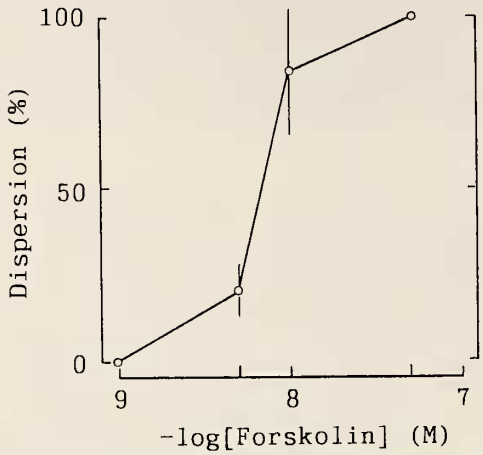

FIG. 8. Relationship between the concentration of forskolin and the magnitude of the pigment-dispersion response of leucophores of *Sebastes inermis*. Each point represents the mean of 10 measurements from three scales. Vertical lines indicate SD.

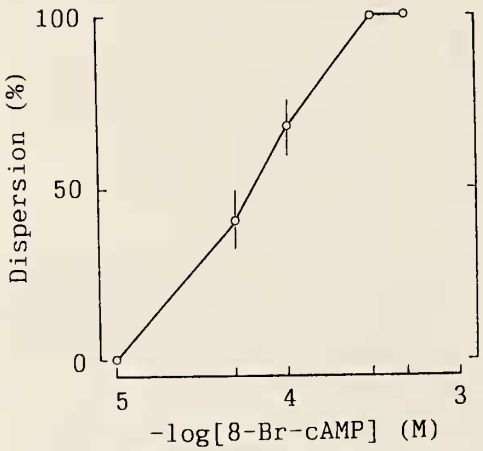

FIG. 9. Relationship between the concentration of 8-Br-cAMP and the magnitude of pigment-dispersion response of leucophores of *Sebastes inermis*. Each point represents the mean of 10 measurements from three scales. Vertical lines indicate SD.

Effects of 8-Br-cAMP

The results of the experiment with forskolin suggested that activation of adenylate cyclase induced the dispersion of pigment. Therefore, the effect of 8-Br-cAMP, an analogue of cAMP, was examined. The drug caused dispersion of pigment. The dispersion response was induced at concentrations above 10^{-5} M and was maximal at 3×10^{-4} M. The concentration-response relationship for the drug is shown in Figure 9. From this Figure, the EC_{50} of 8-Br-cAMP was calculated to be 6.4×10^{-5} M.

Aggregating effects of epinephrine

The results of the experiments with catecholamines suggested that epinephrine might act predominantly on the alpha-adrenoceptors in the pigment-aggregation response, such that the effect of beta receptors might be hidden. This possibility was examined in two series of experiments. First, the effect of epinephrine was examined in the presence of yohimbine. Epinephrine induced pigment dispersion in the presence of yohimbine. The effect was almost the same as that observed with norepinephrine, as was shown in Figure 6. Second, epinephrine was applied to leucophores in

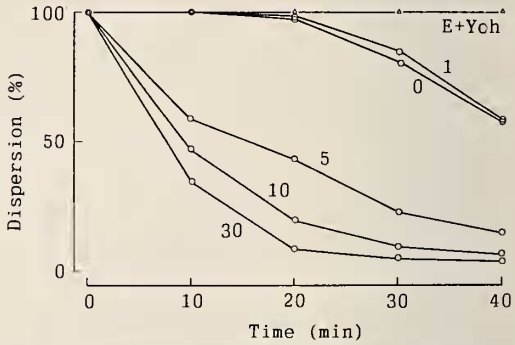

FIG. 10. The pigment-aggregating effects of epinephrine on leucophores of *Sebastes inermis* and inhibition of these effects by yohimbine. Pigment granules in leucophores had been fully dispersed by treatment with K^+-rich saline. The numbers adjacent to the curves indicate the concentrations (μM) of epinephrine. E+Yoh, 10 μM epinephrine in the presence of 50 μM yohimbine. Each point on the graph represents the mean of 10 measurements from three scales.

TABLE 1. Acceleration of the aggregation of pigment in leucophroes of *Sebastes inermis* by 10^{-5} M norepinephrine (NE) in the presence of 5×10^{-5} M propranolol (PRO) and inhibition of the aggregation by 5×10^{-5} M yohimbine (YOH)

Treatment	Half-aggregation time (min) M±SD	No. of measurements (scales)	Level of significance*
Saline	43.0±4.2	10 (3)	
NE+PRO	9.3±1.0	10 (3)	$P<0.001$
NE+PRO+YOH	38.9±5.1	10 (3)	$P>0.2$

* The value shows the significance of the difference in a comparison with the half-aggregation time in saline solution.

a pigment- dispersed state, which had been achieved by 30-min exposure to K^+-rich saline. Epinephrine was effective in accelerating the aggregation of pigment. The effect was concentration-dependent. The aggregating effect was inhibited by yohimbine. These results are illustrated in Figure 10.

Aggregating effects of norepinephrine

Norepinephrine (10^{-5} M) was applied to leucophores in a pigment-dispersed state which had been induced by the K^+-rich saline. When applied with propranolol, norepinephrine accelerated the aggregation of pigment. The effect was antagonized by yohimbine (5×10^{-5} M). These results are given in Table 1, in which the aggregating effect is indicated as the "half-aggregation time" namely, the time that is required for 50% aggregation of pigment.

DISCUSSION

Leucophores do not seem to be common in poikilothermic vertebrates. To date, they have been found only in five species of fish, namely, the killifish *Fundulus heteroclitus* [2, 27] and *F. majalis* [2], the sheepshead minnow *Cyprinodon variegatus* [2], the guppy *Lebistes reticulatus* [30] and the medaka *Oryzias latipes* [23]. As indicated in the Introduction, most physiological information about leucophores has been obtained from *Oryzias latipes*. Thus, further comparative studies on leucophores are clearly desirable.

The leucophores in *Sebastes inermis* appeared yellowish when examined under reflected light, unlike those of *Oryzias latipes* which appeared white. In light-reflecting chromatophores, the coloration of the cell is the net result of the ultrastructure and the composition of pigment organelles. The structure of the pigment organelles differs between leucophores and iridophores: the former contain white granules, while the latter contain iridescent platelets. On the basis of this strictly morphological criterion, it appeared that the chromatophores used in the present studies were a type of leucophores [13]. Details of the ultrastructure will be reported elsewhere.

Leucophores are distributed just under the melanophores in *Fundulus* and *Oryzias*. This arrangement may make their color changes effective since the responses of melanophores to nervous and hormonal stimuli are the opposite of those of leucophores. In *Sebastes inermis,* leucophores appeared characteristically to be randomly distributed without any particular spatial arrangement relative to the other chromatophores.

The rate of translocation of pigment within the cell in response to K^+-rich saline is almost the same in both the leucophores and the melanophores of *Oryzias latipes*: the rate of centrifugal migration in the leucophores being about 30 μm/min [8]. By contrast, in the leucophores of *Sebastes inermis* the rate was so low that it took about 30 min for full dispersion of the pigment in response to stimulation by K^+ ions. The rate was , however, higher than that of dispersion of the motile iridophores in *Odontobutis obscura* [11,

15]. The rate in intracellular migration of organelles may be the net result of effects of their shape and size and the mechano-chemical apparatus responsible for their movements. In this regard, the leucophores of *Sebastes inermis* may be of interest as a material for comparative studies on the movements of chromatophores.

It has been well documented that the aggregation responses of melanophores [3], as well as the dispersion responses of leucophores [8] and also the responses of motile iridophores [15], to K^+ ions are induced *via* the release of transmitter from symphathetic fibers that have been stimulated by K^+ ions. The dispersion response of *Sebastes* leucophores to stimulation by K^+ ions was lost in chemically denervated preparations. This observation indicates that the site of action of K^+ ions for induction of the dispersion of pigment was the nerve fibers. In other words, the leucophores of *Sebastes inermis* are innervated. The effect of K^+ ions was selectively inhibited by propranolol, suggesting that the nerve fibers involved are adrenergic, as are those that innervate to melanophores.

The dispersion responses elicited by isoproterenol and norepinephrine were selectively inhibited by propranolol, indicating that these responses were mediated *via* beta-adrenoceptors in the cells. The effect of forskolin mimicked that of norepinephrine, except that the effect of the former drug was not inhibited by propranolol. These results suggest that an increase in intracellular levels of cAMP induced the dispersion of pigment within the cells. Indeed, 8-Br-cAMP was also effective in inducing the dispersion of pigment. Thus, it was apparent that the adrenergic receptor mechanisms that controlled the dispersion of pigment were of the same type as those in *Oryzias* leucophores [16, 25, 35].

Contrary to our expectation, epinephrine was not effective in inducing the dispersion of pigment in *Sebastes* leucophores. Melanophores responded to epinephrine with a rapid aggregation of melanosomes, as has been observed in the melanophores of many other fish. In the leucophores of *Oryzias,* epinephrine induces dispersion of pigment, even though it is less potent in this regard than norepinephrine [16, 34]. It has been demonstrated that melanophores in various poikilothermic vertebrates, which include fish, frogs, and lizards, possess beta-adrenoceptors that mediate the dispersion of melanosomes [1, 4]. In these melanophores, both norepinephrine and epinephrine are potent inducers of the dispersal of melanosomes [5–7, 10, 24]. In a large variety of tissues and cells other than the chromatophores in vertebrates, these amines act on both alpha- and beta-adrenoceptors as non-specific agonists, although their relative potencies differ from system to system [18, 29, 31–33].

Epinephrine induced the dispersion of pigment after alpha-receptor blockade. This observation suggests that the leucophores had alpha-adrenoceptors in addition to beta-adrenoceptors and that epinephrine might act more effectively at alpha receptors than at beta receptors. This inference is substantiated by the fact that epinephrine acted on the leucophores as a pigment-aggregating agonist when applied to cells in a dispersed state, and the aggregating effect was inhibited by yohimbine. Norepinephrine also induced aggregation of pigment in the presence of propranolol and the effect was blocked by yohimbine. These results demonstrate that the aggregating response was induced *via* alpha-adrenoceptors. In leucophores of *Oryzias latipes,* the existence of alpha-adrenoceptors that participate in the aggregation of pigment has also been demonstrated [9].

The results of the present study indicate that the adrenoceptor mechanisms involved in the migration of pigment in the leucophores of *Sebastes inermis* are fundamentally the same as those in *Oryzias latipes.* The "novel" chromatophores in *Sebastes inermis* are physiologically, as well as morphologically, a type of leucophore.

REFERENCES

1 Bagnara JT, Hadley ME (1973) Chromatophores and Color Change. Prentice-Hall Inc, Englewood Cliffs, New Jersey, pp 133–144

2 Fries EFB (1942) White pigmentary effectors (leucophores) in killifish. Proc Natl Acad Sci 28: 396–401

3 Fujii R (1959) Mechanisms of ionic action in the melanophore system of fish I. Melanophore-concentrating action of potassium and some other

ions. Annot Zool Jpn 32: 47–58

4 Fujii R, Oshima N (1986) Control of chromatophore movements in teleost fishes. Zool Sci 3: 13–47

5 Goldman JM, Hadley ME (1969) The beta adrenergic receptor and cyclic 3′,5′-adenosine monophosphate: Possible roles in the regulation of melanophores responses of the spadefoot toad, *Scaphiopus couchi*. Gen Comp Endocrinol 13: 151–163

6 Goldman JM, Hadley ME (1969) *In vitro* demonstration of adrenergic receptors controlling melanophore responses of the lizard, *Anolis carolinensis*. J. Pharmacol Exp Ther 166: 1–7

7 Hadley ME, Goldman JM (1970) Adrenergic receptors and geographic variation in *Rana pipiens* chromatophore responses. Am J Physiol 219: 72–77

8 Iga T (1978) The mode of action of potassium ions on the leukophores of a fraeshwater teleost, *Oryzias latipes*. J. Exp Zool 205: 413–422

9 Iga T (1979) Alpha adrenoceptors: Pigment aggregation in *Oryzias* leucophores. Mem Fac Sci Shimane Univ 13: 87–95

10 Iga T (1983) Basic properties of beta adrenergic receptors mediating melanosome dispersion of melanophores in a cyprinid fish, *Zacco temmincki*. Comp Biochem Physiol 76C: 297–303

11 Iga T, Kinutani J, Maeno N (1990) Motility of cultured iridophores from the freshwater goby *Odontobutis obscura*. Zool Sci 7: 401–407

12 Iga T, Matsuno A (1986) Motile iridophores of a freshwater goby, *Odontobutis obscura*. Cell Tissue Res 244: 165–171

13 Iga T, Matsuno (1992) A novel leucophore in the darkbanded rockfish, *Sebastes inermis*. Zool Sci 6: 1226

14 Iga T, Takabatake I (1982) Denervated melanophores of the dark chub *Zacco temmincski*: Method of denervation and the evaluation of preparations for physiological experiments. Annot Zool Jpn 55: 61–69

15 Iga T, Takabatake I, Watanabe S (1987) Nervous regulation of motile iridophores of a freshwater goby, *Odontobutis obscura*. Comp Biochem Physiol 88C: 319–314

16 Iga T, Yamada K, Iwakiri M (1977) Adrenergic receptors mediating pigment dispersion in leucophores of a teleost, *Oryzias latipes*. Mem Fac Lit Sci Shimane Univ (Nat Sci) 11: 63–72

17 Kamei-Takeuchi I, Eguchi G, Hama T (1968) Ultrastructure of the pteridine pigment granules of the larval xanthophore and leucophore in *Oryzias latipes* (Teleostean fish). Proc Japan Acad 44: 959–963

18 Lands AM, Arnold A, McAuliff JP, Luduena FP, Brown TG (1967) Differentiation of receptor systems activated by sympathomimetic amines. Nature 214: 597–598.

19 Lythgoe JN, Shand J (1982) Changes in spectral reflexions from the iridophores of the neon tetra. J Physol 325: 23–34

20 Maeno N, Iga T (1992) Adrenergic mechanisms associated with the movement of platelets in iridophores from the freshwater goby, *Odontobutis obscura*. Comp Biochem Physiol 102C: 233–237

21 Matsuno A, Iga T (1989) Ultrastructural observations of motile iridophores from the freshwater goby, *Odontobutis obscura*. Pigment Cell Res 2: 431–438

22 Menter DG, Obika M, Tchen TT, Taylor JD (1979) Leucophores and iridophores of *Fundulus heteroclitus*: Biophysical and ultrastructural properties. J Exp Zool 160: 103–120

23 Miyoshi S (1952) Response of iridocytes in isolated scales of the medaka (*Oryzias latipes*) to chlorides. Annot Zool Jpn 25: 21–29

24 Morishita F (1987) Responses of the melanophores of the medaka, *Oryzais latipes*, to adrenergic drugs: Evidence for involvement of alpha$_2$ adrenergic receptors mediating melanin aggregation. Comp Biochem Physiol 88C: 69–74

25 Namoto S, Yamada K (1987) Effects of forskolin, isoproterenol, and lithium ions on leucophores of a teleost, *Oryzias latipes*: Evidence for involvement of adenylate cyclase in pigment-dispersion response. Comp Biochem Physiol 86C: 91–95

26 Obika M (1988) Ultrastructure and physiological responses of leucophores of medaka *Oryzias latipes*. Zool Sci 5: 311–321

27 Odiorne JM (1933) The occurrence of guanophores in *Fundulus*. Proc Natl Acad Sci 19: 750–754

28 Oshima N, Sato M, Kumazawa T, Okeda N, Kasukawa H, Fujii R (1985) Motile iridophores play the leading role in damselfish coloration. In "Pigment Cell 1985" Ed by J Baganara, SN Klaus, E Paul, M Schartl, Univ Tokyo Press, Tokyo, pp 241–246

29 Stene-Larsen G (1981) Comparative aspects of cardiac adrenoceptors: Characterization of b$_2$-adrenoceptor as a common "adrenaline"-receptor in vertebrate heart. Comp Biochem Physiol 70C: 1–12

30 Takeuchi I (1976) Electron microscopy of two types of reflecting chromatophores (iridophores and leucophores) in the guppy, *Lebistes reticulatus* Peters. Cell Tissue Res 173: 17–27

31 Tetens V, Lykkeboe G (1988) Potency of adrenaline and noradrenaline for red cells of rainbow trout, *Salmo gairdneri*. J Exp Biol 134: 267–280

32 William LT, Jarett L, Lefkowitz RJ (1976) Adipocyte β-adrenergic receptors. J Biol Chem 251: 3096–3104

33 Williams LT, Mullikin D, Lefkowitz RJ (1976) Identification of α-adrenergic receptors in uterine

smooth muscle membrane by [^{3}H] dihydroergocryptine binding. J Biol Chem 251: 6915–6923

34 Yamada K (1980) Actions of sympathomimetic amines on leucophores in isolated scales of a teleost fish with special reference to beta-adrenoceptors mediating pigment dispersion. J Sci Hiroshima Univ B (1) 28: 95–114

35 Yamada K, Iwakiri M (1982) Effects of cyclic-AMP, methylxanthines and imidazole on fish leucophores. Annot Zool Jpn 55: 199–209